

=> d his

(FILE 'HOME' ENTERED AT 19:28:57 ON 30 JUL 2007)
FILE 'CA' ENTERED AT 19:29:07 ON 30 JUL 2007
L1 2918 S (CAMP(2A) RESPONSIVE(2A) ELEMENT(2A) BINDING OR CREB) AND KINASE
L2 8 S L1 AND QUENCH?
L3 81 S L1 AND FLUORES?
L4 11 S L2-3 AND PY<2000
L5 32 S L2-3 AND PATENT/DT
FILE 'BIOSIS' ENTERED AT 19:37:38 ON 30 JUL 2007
L6 10 S L4
FILE 'MEDLINE' ENTERED AT 19:38:02 ON 30 JUL 2007
L7 17 S L4
FILE 'CA, BIOSIS, MEDLINE' ENTERED AT 19:39:33 ON 30 JUL 2007
L8 52 DUP REM L4 L5 L6 L7 (18 DUPLICATES REMOVED)

=> d bib,ab 18 1-52

L8 ANSWER 44 OF 52 CA COPYRIGHT 2007 ACS on STN
AN 125:27565 CA
TI Analysis of the structural properties of **cAMP-responsive element-binding** protein (**CREB**) and phosphorylated **CREB**
AU Richards, Jane P.; Bachinger, Hans Peter; Goodman, Richard H.; Brennan, Richard G.
CS Dep. Biochem. Mol. Biol., Oregon Health Sci. Univ., Portland, OR, 97201, USA
SO Journal of Biological Chemistry (1996), 271(23), 13716-13723
AB The transcription factor **CREB** (**cAMP responsive element binding** protein) is activated by protein **kinase** A (PKA) phosphorylation of a single serine residue. To investigate possible mechanisms of **CREB** regulation by phosphorylation, we initiated a structural and biophys. characterization of the full-length, wild-type **CREB** protein, an altered **CREB** protein (**CREB/SER**) in which the three cysteine residues in the DNA-binding domain were replaced with serine residues and a truncated protein (ACT265) which encompasses the entire activation domain of **CREB**. CD reveals that **CREB** and **CREB/SER** have identical secondary structures and contain approx. 20% α -helix, 9% β -strand, 34% β -turn, and 37% random coil structures. PKA phosphorylation does not alter the CD spectra, and bound to DNA. Protease cleavage patterns indicate that PKA phosphorylation does not induce a global conformational change in **CREB**. Furthermore, PKA phosphorylation does not change the DNA binding affinity of **CREB** for either canonical or non-canonical CRE sequences as measured by a **fluorescence** anisotropy DNA binding assay. Since PKA phosphorylation of **CREB** results in its specific binding to the transcriptional coactivators **CREB**-binding protein and p300, we suggest that the PKA activation of **CREB** occurs by the prodn. of specific, complementary interactions with these proteins, rather than through the previously proposed mechanisms of a phosphorylation-dependent conformational change or increased DNA binding affinity.

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STN INTERNATIONAL LOGOFF AT 19:40:21 ON 30 JUL 2007